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The relationship between prolactin level and inflammatory markers in women with polycystic ovary syndrome

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ABSTRACT

Aims: This study aimed to investigate the relationship between prolactin (PRL) levels and inflammatory markers in women with polycystic ovary syndrome (PCOS) compared to controls.

Methods: A total of 120 women, 60 with PCOS and 60 controls, were included in this study. The participants were divided into four groups based on their PRL levels: Hyperprolactinemia (HPRL) PCOS, normal PRL PCOS, HPRL controls, and normal PRL controls. The levels of various inflammatory markers, including C-reactive protein (CRP), Hemoglobin (Hb), red cell distribution width (RDW-SD), mean platelet volume (MPV), and platelet distribution width (PDW), were measured and compared between the groups. The correlation between PRL levels and inflammatory markers was also analyzed.

Results: The results show that the mean value of CRP was higher in the PCOS group with HPRL (2.15±1.61) compared to the PCOS group with normal PRL levels (1.68±1.19), but the difference was not statistically significant (p=0.432).The correlation results show that there was a statistically significant negative correlation between PRL levels and Hb (r=-0.313, p=0.015) and a positive correlation between PRL levels and RDW-SD (r=0.352, p=0.006) in PCOS patients.In PCOS patients, higher PRL levels were associated with increased RDW-SD, decreased Hb levels, decreased platelet count, and increased MPV and PDW. However, there was no significant correlation between PRL levels and inflammatory markers in the control group.

Conclusion: The results of this study suggest that higher PRL levels may be associated with increased inflammation in PCOS patients. However, further research is needed to understand the underlying mechanisms and potential clinical implications of these associations.

Keywords: Polycystic ovary syndrome, prolactin, inflammatory markers, inflammatory markers, PCOS

INTRODUCTION

Women in their reproductive age are often impacted by polycystic ovary syndrome (PCOS), a widespread endocrine condition.[1]Hyperprolactinemia (HPRL) is an endocrine disorder in which the bloodstream contains excessive amounts of prolactin (PRL), as seen in its distinguishing feature. PRL is a hormone responsible for lactation, breast development, and other actions needed for normal physiology.[2]

On the other hand, chronic inflammation is a common feature in both PCOS and HPRL. In PCOS, elevated levels of inflammation markers, such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α), have been observed.[3,4] Similarly, HPRL has been characterized by elevated levels of inflammatory markers.[2] The relationship between HPRL and PCOS may be mediated by several interconnected mechanisms, including inflammation, immune system modulation, insulin resistance and gut microbiota alterations.[5]

Given the evidence connecting prolactin, inflammation, and PCOS, it is important to understand their interrelationships. Elevated PRL levels could contribute to chronic inflammation by promoting immune cell activation or cytokine release from adipose tissue.[6] Therefore, it is plausible that there may be a relationship between PRL levels and markers of inflammation in PCOS patients. The mechanisms underlying this association remain unclear, but investigating their potential interplay could provide new perspectives into the development of this multifaceted disorder.

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Some studies have found higher levels of inflammatory markers like CRP, TNF-alpha, and IL-6 in women with HPRL and PCOS compared to controls.[7,8] Given the evidence connecting prolactin, inflammation, and PCOS, it is important to understand their interrelationships.Elevated PRL levels could contribute to chronic inflammation by promoting immune cell activation or cytokine release from adipose tissue.[9,10] Therefore, it is plausible that there may be a relationship between PRL levels and markers of inflammation in PCOS patients.

The mechanisms underlying this association remain unclear, but investigating their potential interplay could provide new perspectives into the development of this multifaceted disorder.In this study, we focus on the inflammatory effect of prolactin and its captivating dimension to the research. We aim to shed light on the potential interplay between PRL levels and markers of inflammation in PCOS patients, with a particular focus on the inflammatory effect of PRL. By investigating this relationship, we hope to provide new insights into the development of PCOS and its potential clinical implications.

METHODS

This retrospective study was conducted between June 2021 and December 2022 at Bezmialem Vakıf University Hospital Gynecology and Obstetric Department. The study was carried out with the permission of Bezmialem Vakıf University Hospital Ethics Committee (Date: 07.06.2023, Decision No:2023/190). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

A total of 120 patients aged 18-30 who applied to our outpatient clinics with menstrual irregularity with PCOS, and a control group with similar age and body mass index (BMI) were included in the study. For determining the eligibility criteria for the study, based on the history, physical examination, and ultrasonography evaluation taken from the patients who applied to the outpatient clinic between the specified dates, the diagnosis of PCOS was made on the condition of having at least two of the 2003 Rotterdam Consensus criteria.[11]

Inclusion criteria in the study group were: (1) women between the ages of 18-30 who cannot have children despite wanting a child for at least one year; (2) those diagnosed with PCOS, provided that they have at least two of the 2003 Rotterdam Consensus criteria; (3) those who are not diagnosed with diabetes mellitus, impaired glucose tolerance, thyroid dysfunction, HPRL, and hypercortisolism; (4) women were not given oral contraceptives or any medication known to alter insulin metabolism, lipid, or hormones in the last 90 days; (5) non-smokers.

Criteria for exclusion from the study group were: (1) Smokers; (2) women with diabetes, hypertension, and endocrinopathy; (3) individuals who have consumed medication for the treatment of PCOS, and those who have taken oral contraceptives within the past three months., those who use drugs that increase insulin sensitivity or those who use drugs for hyperlipidemia; (4) patients taking vitamin B12 supplements were excluded due to the effect of B12 on reducing homocysteine, which could confound results. In this study, four groups are considered.

Group I is defined as follows: women with PCOS with HPRL.

Group II is defined as follows: women with PCOS with normal PRL.

Group III is defined as follows: healthy women with HPRL.

Group IV is defined as follows: healthy women with normal PRL.

Among the patients who applied to our routine obstetrics and gynecology outpatient clinic with menstrual irregularity and a desire to have children, we examined the patients on the third day of their menstrual cycle. Levels of inflammatory markers including CRP, TNF-α, and IL-6 were examined. The data of PCOS patients and the control group were analyzed retrospectively.

Statistical Analysis

The sample size of 120 was determined based on a power analysis indicating this would provide 80% power to detect significant differences between groups. Statistical analysis was conducted using SPSS version 26**.** Normality of data was assessed using the Shapiro-Wilk test. Differences between groups were analyzed using independent t-tests for normal data and Mann-Whitney U tests for non-normal data. Associations were examined using Spearman correlation coefficients. $P<0.05$ will be considered statistically significant.

RESULTS

This study included 120 age-matched (24.83±2.27) and body mass index (BMI)-matched (23.93±1.09) women. Table 1 shows the relationship between case and control groups regarding inflammatory markers. In the following, inflammatory markers of women with PCOS in two groups (HPRL and normal PRL) were examined. As stated in Table 1, an Independent t-test showed a significant statistical association between group I and II regarding Hemoglobin (Hb) ($p<0.05$). Hb in PCOS patients was significantly elevated in the normal PRL group. As stated in Table 1, a Mann-Whitney U test found a statistically significant association between groups I and II regardingred cell distribution width (RDW-SD) and mean platelet volume (MPV) levels ($p<0.05$). There was a significant reduction in these parameters in the normal PRL group. There was a significant association between group I and II regarding platelets ($p<0.05$).

The results show a statistically significant difference in PRL levels between PCOS patients and controls, with PCOS patients having higher levels of PRL. However, there is no statistically significant difference in CRP levels between the PCOS and control groups. Hb levels are significantly lower in PCOS patients with HPRL compared to controls with normal PRL levels. RDW-SD is significantly higher in PCOS patients with HPRL compared to controls with normal PRL levels. Platelet levels are significantly lower in

Table 1. The relationship between case and control groups in terms of inflammatory markers

	PCOS (n=60) Mean±SD			Controls (n=60) Mean±SD			
Parameter	**Hyper-PRL (Group I) n=30**	**Normal-PRL (Group II) n=30**	**p**	**Hyper-PRL (Group III) n=30**	**Normal-PRL (Group IV) n=30**	**p**	**p**
PRL	31.13±2.62	14.87±2.92	**<0.001**	31.4±2.82	14.77±3.24	**<0.001**	0.759
CRP	2.15±1.61	1.68±1.19	0.432	2.07±1.73	1.27±0.83	0.086	0.782
Hb	12.31±1.39	13.16±0.99	**0.008**	12.18±1.28	12.84±1.21	0.045	0.695
RDW-SD	35.28±3.08	33.53±3.5	**<0.001**	34.75±2.6	34.02±2.91	**0.041**	0.589
Platelet	241433.33±56159.68	288300±69559.77	**0.010**	242133.33±56617.13	278566.67±67133.26	**0.041**	0.988
MPV	9.77±1.55	9±2.09	**0.019**	9.63±1.61	9.65±1.18	0.336	0.711
PDW	13.39±3.48	12.39±4.53	0.105	13.61±3.69	11.64±3.06	**0.008**	0.900

*** SD, standard deviation; PLR, platelet/lymphocyte ratio; CRP, C-reactive protein; Hb, Hemoglobin; RDW-SD, a red cell distribution width; MPV, mean platelet volume; PDW, platelet distribution width;. Parametric values were expressed as means ± SD.**

controls with HPRL compared to controls with normal PRL levels. MPV is significantly higher in PCOS patients with HPRL compared to controls with normal PRL levels. platelet distribution width (PDW) is significantly higher in controls with HPRL compared to PCOS patients with normal PRL levels.

In PCOS patients, there is a statistically significant positive correlation between PRL levels and RDW-SD (r=0.352, p=0.006). There is a statistically significant negative correlation between PRL levels and Hb (r=-0.313, p=0.015). There is a statistically significant negative correlation between PRL levels and platelet count (r=-0.293, p=0.023). There is a statistically significant positive correlation between PRL levels and MPV (r=0.300, p=0.020). There is a statistically significant positive correlation between PRL levels and PDW (r=0.287, p=0.026).

In controls, there is no statistically significant correlation between PRL levels and any of the inflammatory markers.

Based on these results, we can conclude that in PCOS patients, higher PRL levels are associated with increased RDW-SD, decreased Hb levels, decreased platelet count, and increased MPV and PDW. However, there is no significant correlation between PRL levels and inflammatory markers in the control group.

It is important to note that these results are based on the correlation analysis and do not establish a causal relationship between PRL levels and inflammatory markers. Further research is needed to understand these associations underlying mechanisms and potential clinical implications.

Table 2. Correlation between PRL and Inflammatory Markers

	PCOS		Controls		
Parameter	**r**	**p-values**	**Parameter**	**r**	**p-values**
AMH (ng/ml)	-0.057	0.668	AMH (ng/ml)	-0.050	0.704
FSH (mIU/ml)	0.047	0.719	FSH (mIU/ml)	-0.110	0.404
LH (mIU/ml)	-0.083	0.531	LH (mIU/ml)	0.011	0.936
E2 (pg/ml)	-0.013	0.923	E2 (pg/ml)	0.046	0.730
TSH (mIU/l)	-0.066	0.615	TSH (mIU/l)	0.116	0.376
Free T4 (pmol/l)	-0.090	0.493	Free T4 (pmol/l)	0.011	0.934
CRP (mg/dl)	0.082	0.536	CRP (mg/dl)	0.199	0.128
Leukocyte	0.205	0.115	Leukocyte	0.006	0.964
Neutrophil	-0.063	0.633	Neutrophil	-0.017	0.900
Lymphocyte	-0.126	0.339	Lymphocyte	0.103	0.435
Monocyte	-0.037	0.777	Monocyte	0.004	0.974
Basophil	-0.131	0.317	Basophil	0.021	0.874
Hb	-.313*	**0.015**	Hemoglobin	-0.176	0.178
RDW-SD	.352**	**0.006**	RDW-SD	0.211	0.106
Platelet	-.293*	**0.023**	Platelet	-.304*	**0.018**
MPV	.300*	**0.020**	MPV	0.076	0.562
PDW	0.147	0.261	PDW	.287*	**0.026**
NLO	0.072	0.583	NLO	-.043	0.746
LMO	-0.079	0.546	LMO	0.038	0.776
TLO	-0.013	0.920	TLO	-0.308	**0.017**

*p<0.05: statistically significant.
*AMH, Anti-Müllerian hormone; FSH, follicle-stimulating hormone; LH, luteinizing hormone; E2, estradiol; TSH, thyroid stimulating hormone; Free T4, free thyroxin; CRP, C-reactive protein; Hb, Hemoglobin; RDW-SD, a red cell distribution width; MPV, mean platelet volume; PDW, platelet distribution width; NLR,neutrophil/lymphocyte ratio; LMR, lymphocyte-to-monocyte ratio; PLR, platelet/lymphocyte ratio. Parametric values were expressed as means ± SD. SD, standard deviation.

DISCUSSION

The present study aimed to investigate the relationship between PRL levels and inflammatory markers in PCOS patients and controls. Results showed that PCOS patients had significantly higher levels of PRL than controls. However, there was no significant difference in creative protein (CRP) levels between the two groups. Hb levels were significantly lower in PCOS patients with HPRL compared to controls with normal PRL levels. RDW-SD was significantly higher in PCOS patients with HPRL compared to controls with normal PRL levels. Platelet levels were significantly lower in controls with HPRL compared to controls with normal PRL levels. MPV was significantly higher in PCOS patients with HPRL compared to controls with normal PRL levels. PDW was significantly higher in controls with HPRL compared to PCOS patients with normal PRL levels.

The results of this study are consistent with previous research that has shown that women with PCOS have elevated levels of inflammatory markers such as CRP.[12] However, our study did not find a significant difference in CRP levels between PCOS patients and controls. This inconsistency may be due to differences in the study population, sample size, and measurement methods. In addition, our study found that Hb levels were significantly lower in PCOS patients with HPRL compared to controls with normal PRL levels. This finding is consistent with previous research that has shown that PCOS patients have higher Hb levels than controls.[13] The reason for this association is not clear, but it may be related to the hormonal imbalances that occur in PCOS.

The correlation between PRL levels and various inflammatory markers in PCOS patients and controls in our study found that in PCOS patients, higher PRL levels were associated with increased RDW-SD, decreased Hb levels, decreased platelet count, and increased MPV and PDW. However, there was no significant correlation between PRL levels and inflammatory markers in the control group. These results suggest that higher PRL levels may be associated with increased inflammation in PCOS patients.

Our findings are consistent with previous research that has shown a link between PRL levels and platelet activation in women with PCOS.[14] The present study found that higher PRL levels were associated with increased MPV and PDW in PCOS patients. This finding is consistent with previous research that has shown that women with PCOS have higher MPV levels than women without PCOS.[15] The present study also found that higher PRL levels were associated with decreased platelet count in PCOS patients. This finding is consistent with previous research that has shown that women with PCOS have lower platelet counts than women without PCOS.[16]

The hypothesis of this study was that higher PRL levels would be associated with increased inflammatory markers in PCOS patients compared to controls. The results of this study partially support the hypothesis that higher PRL levels are associated with increased inflammatory markers in PCOS patients. Specifically, higher PRL levels were associated with increased RDW-SD, decreased Hb levels, decreased platelet count, and increased MPV and PDW in PCOS patients. However, there was no significant correlation between PRL levels and inflammatory markers in the control group.

Similar studies have shown inconsistent results regarding the association between PRL levels and inflammatory markers in PCOS patients. For example, a study by Rudnicka et al.[12] found increased serum CRP in women with PCOS compared to healthy controls. However, our study did not find a significant difference in CRP levels between PCOS patients and controls. Another study by Saei Ghare Nazet al.[17] found a link between PRL levels and platelet activation in women with PCOSwhich is consistent with our finding that higher PRL levels were associated with decreased platelet count in PCOS patients.

The findings of this study have several implications for the understanding and management of PCOS and its associated inflammatory markers.Higher PRL levels may be associated with increased inflammation in PCOS patients. Specifically, higher PRL levels were associated with increased RDW-SD, decreased Hb levels, decreased platelet count, and increased MPV and PDW in PCOS patients. The lack of significant correlation between PRL levels and inflammatory markers in the control group suggests that the observed associations are specific to PCOS patients. This highlights the importance of considering the unique pathophysiology of PCOS when investigating the relationship between hormonal imbalances and inflammatory markers.

The significant differences in Hb, RDW-SD, platelet count, MPV, and PDW between PCOS patients and controls suggest that these inflammatory markers may be useful in the diagnosis and management of PCOS. Further research is needed to determine the clinical implications of these findings.

The lack of significant difference in CRP levels between PCOS patients and controls suggests that CRP may not be a reliable marker of inflammation in PCOS. Other inflammatory markers, such as RDW-SD, MPV, and PDW, may be more useful in this context. The significant correlation between PRL levels and RDW-SD, MPV, and PDW in PCOS patients suggests that these markers may be useful in monitoring the effects of PRL-lowering therapies in PCOS patients.

Overall, these findings suggest that PRL may play a role in the inflammatory processes associated with PCOS, and that inflammatory markers such as RDW-SD, MPV, and PDW may be useful in the diagnosis and management of PCOS. Further research is needed to determine the clinical implications of these findings and to investigate the underlying mechanisms of the observed associations.

Limitations of the Study

The present study has several limitations that should be considered when interpreting the results. Firstly, the retrospective design of the study may have introduced bias into the data collection process. This design relies on data that has already been collected, which may limit the ability to control for confounding variables. Secondly, the sample size of the study is relatively small, with only 60 PCOS patients and 60 controls. This may limit the generalizability of the findings and may reduce the statistical power of the study.

Thirdly, the study only measured a limited number of inflammatory markers, including CRP, Hb, RDW-SD, MPV, and PDW. Other markers, such as interleukins and tumor necrosis factor, may also be relevant to the inflammatory processes associated with PCOS. Therefore, the findings

of this study may not be representative of the full range of inflammatory markers that are associated with PCOS.

Finally, PRL levels can vary widely depending on a variety of factors, including stress, exercise, and medication use. This variability may limit the ability to draw firm conclusions about the relationship between PRL levels and inflammatory markers. Therefore, further research is needed to confirm the findings of this study and to investigate the underlying mechanisms of the observed associations.

Despite these limitations, the findings of this study have important implications for the understanding and management of PCOS and its associated inflammatory markers. The study found that higher PRL levels were associated with increased inflammation in PCOS patients, and that inflammatory markers such as RDW-SD, MPV, and PDW may be useful in the diagnosis and management of PCOS. However, further research is needed to confirm these findings and to investigate the underlying mechanisms of the observed associations.

CONCLUSION

Our study found that higher PRL levels were associated with increased inflammation in PCOS patients. Specifically, higher PRL levels were associated with increased RDW-SD, decreased Hb levels, decreased platelet count, and increased MPV and PDW in PCOS patients. These findings suggest that PRL may play a role in the development of inflammation in PCOS patients. However, further research is needed to understand the underlying mechanisms and potential clinical implications of these associations.

ETHICAL DECLARATIONS

Ethics Committee Approval: The study was carried out with the permission of Bezmialem Vakıf University Hospital Ethics Committee (Date: 07.06.2023, Decision No:2023/190).

Informed Consent: Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process: Externally peer-reviewed.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

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Author Contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper and that they have approved the final version.

REFERENCES

1. Alanya Tosun Ş, Gurbuz T, Cebi A, Tosun A, Gokmen O, Usta M. Association of increased levels of omentin-1 and carotid intima-media thickness with early signs of cardiovascular risk in patients with polycystic ovary syndrome: a prospective case control study. *J Obstet Gynaecol Res.* 2022;48(1):169-177. doi:10.1111/jog.15077
2. Asfuroğlu Y, Kan Ö, Asfuroğlu M, Baser E. Association between dry eye and polycystic ovary syndrome: subclinical inflammation may be part of the process. *Eye Contact Lens.* 2021;47(1):27-31. doi:10.1097/ICL.0000000000000716
3. Barbakadze L, Kristasashvili J. Antimullerian hormone in cases of different reproductive pathologies. *Georgian Med News.* 2014;(232-233):16-21.
4. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. *Fertil Steril.* 2004;81(1):19-25. doi:10.1016/j.fertnstert.2003.10.004
5. Dokuzeylül Güngör N, Güngör K, Yurci A, Cil K, Hatırnaz Ş. Ovarian drilling down-regulates endometrial nuclear factor-κB p65 expression in women with PCOS: a prospective case-control study. *Turk J Obstet Gynecol.* 2022;19(1):45-50. doi:10.4274/tjod.galenos.2022.44845
6. Gungor ND, Gurbuz T. Pregnancy outcomes of intrauterine insemination in age-matched young women according to serum anti-mullerian hormone levels. *JRM.* 2021;66:195-202.
7. Güngör ND, Gürbüz T. Prediction of the number of oocytes based on AMH and FSH levels in IVF candidates. *J Surg Med.* 2020;4:733-737. doi:10.28982/josam.759207.
8. Gürbüz T, Gökmen O, Demirçivi Bör E, Uluocak A. Investigating the relationship of serum levels of afamin and interleukin-10 with insulin resistance in infertile women with polycystic ovary syndrome. *J Surg Med.* 2021;5(3):264-268. doi:10.28982/josam.874039.
9. Gürbüz T, Gökmen O, Dokuzeylül Güngör N. Polikistik over sendromu bulunan kadınlarda glikoz potasyum oranının tanısal değerinin insülin ile karşılaştırılması . *Cukurova Med J.* 2021;46(1):381-386. doi.org/10.17826/cumj.782931
10. Gurbuz T, Gokmen O. A Novel Index to overcome the assessment limitations of insulin resistance in polycystic ovary syndrome homeostatic model assessment index to red blood cell distribution width ratio. *JRM.* 2021;66(1-2):9-14.
11. Overgaard M, Glintborg D, Christesen HT, Jensen TK, Andersen MS. Maternal prolactin is associated with glucose status and PCOS in pregnancy: odense child cohort. *Eur J Endocrinol.* 2020;183(3):307-316. doi:10.1530/EJE-20-0144
12. Rudnicka E, Kunicki M, Suchta K, Machura P, Grymowicz M, Smolarczyk R. Inflammatory markers in women with polycystic ovary syndrome. *Biomed Res Int.* 2020;2020:4092470. doi:10.1155/2020/4092470
13. Han Y, Kim HS, Lee HJ, Oh JY, Sung YA. Metabolic effects of polycystic ovary syndrome in adolescents. *Ann Pediatr Endocrinol Metab.* 2015;20(3):136-142. doi:10.6065/apem.2015.20.3.136
14. Yilmaz Ö, Calan M, Kume T, Temur M, Yesil P, Senses MY. The effect of prolactin levels on MPV in women with PCOS. *Clin Endocrinol (Oxf).* 2015;82(5):747-752. doi:10.1111/cen.12647
15. Li L, Yu J, Zhou Z. Mean platelet volume and polycystic ovary syndrome: a systematic review and meta-analysis. *J Int Med Res.* 2022;50(1):3000605211067316. doi:10.1177/03000605211067316
16. Yilmaz Ö, Mehmet C, Kelekci S, Temur M. Association between red blood cell distribution width and polycystic ovary syndrome. *Endocr Res.* 2015;40(4):181-187. doi:10.3109/07435800.2014.987398
17. Saei Ghare Naz M, Mousavi M, Mahboobifard F, Niknam A, Ramezani Tehrani F. A meta-analysis of observational studies on prolactin levels in women with polycystic ovary syndrome. *Diagnostics (Basel).* 2022;12(12):2924. doi:10.3390/diagnostics12122924

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